(Reprinted from the Birmingham Medical Review, Dec., 1891.)

REDUPLICATION OF THE SECOND SOUND OF THE HEART.

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SINCE it was discovered by Bouillaud,* this symptom has, with almost perfect unanimity, been ascribed to asynchronous closure, or tension, of the aortic and pulmonary valves. When we enquire more closely, however, into the views held by different observers as to the means by which this failure to act simultaneously is produced, we find considerable divergence of opinion. By von Bamberger † it was attributed to an irregular contraction of the arterial walls, causing the propulsion of successive blood-waves towards the sigmoid valves. Potain ‡ seems to have been the first to assume a difference of pressure within the aorta and pulmonary artery as the cause of the asynchronous action of the valves, and his interpretation has, with various modifications, remained, to some extent, valid until the present time.

It was remarked above that there has been an almost perfect

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* Traité Clinique des Maladies du Cœur, Tome i., p. 189. 1835.
† Lehrbuch der Krankheiten des Herzens, s. 73. 1857.
‡ L'Union Médicale, N.S. Tome xxxi., pp. 307. 357, 438, 595, 611. 1866.

unanimity in regard to the essential cause of the double sound, but it must not be forgotten that there have been other explanations. The only theory worthy of mention which refers the phenomenon to causes other than asynchronous action of the sigmoid valves is that propounded by Sansom.* He considers that the second part of the double sound is produced by the sudden tension of the abnormal mitral curtains, consequent upon the relaxation of the left ventricle. Even if this theory were sufficient to explain the double second sound in cases of mitral disease, it would not account for physiological doubling; and Sansom allows that there are cases which can be best explained by asynchronism of the two second sounds.

In truth, the mere existence of the doubling caused by physiological acts, such as holding the breath after a deep inspiration, is sufficient to prove that slight alterations of the relative pressure in the different parts of the central apparatus are sufficient to produce a double second sound. And if double second sound can be produced by slight changes of relative pressure, it is unnecessary to go further in search of a cause for the persistent double second sound of mitral disease. The manner in which changes of relative pressure effect the result must, as is generally recognised, be by causing the systole of one ventricle to terminate a little later than that of the other. The main object of this communication is to attempt to make this matter somewhat clearer.

Long-continued study of some tracings obtained in Birmingham from the conus arteriosus of a man presenting a sternal fissure has made the subject stand out very clearly in my own mind, since they furnish a graphic record of the relations in which cessation of ventricular systole and production of second sound stand to each other. The case has been fully described by Malet and myself,† and some of the tracings further analysed by me,‡ in previous papers.

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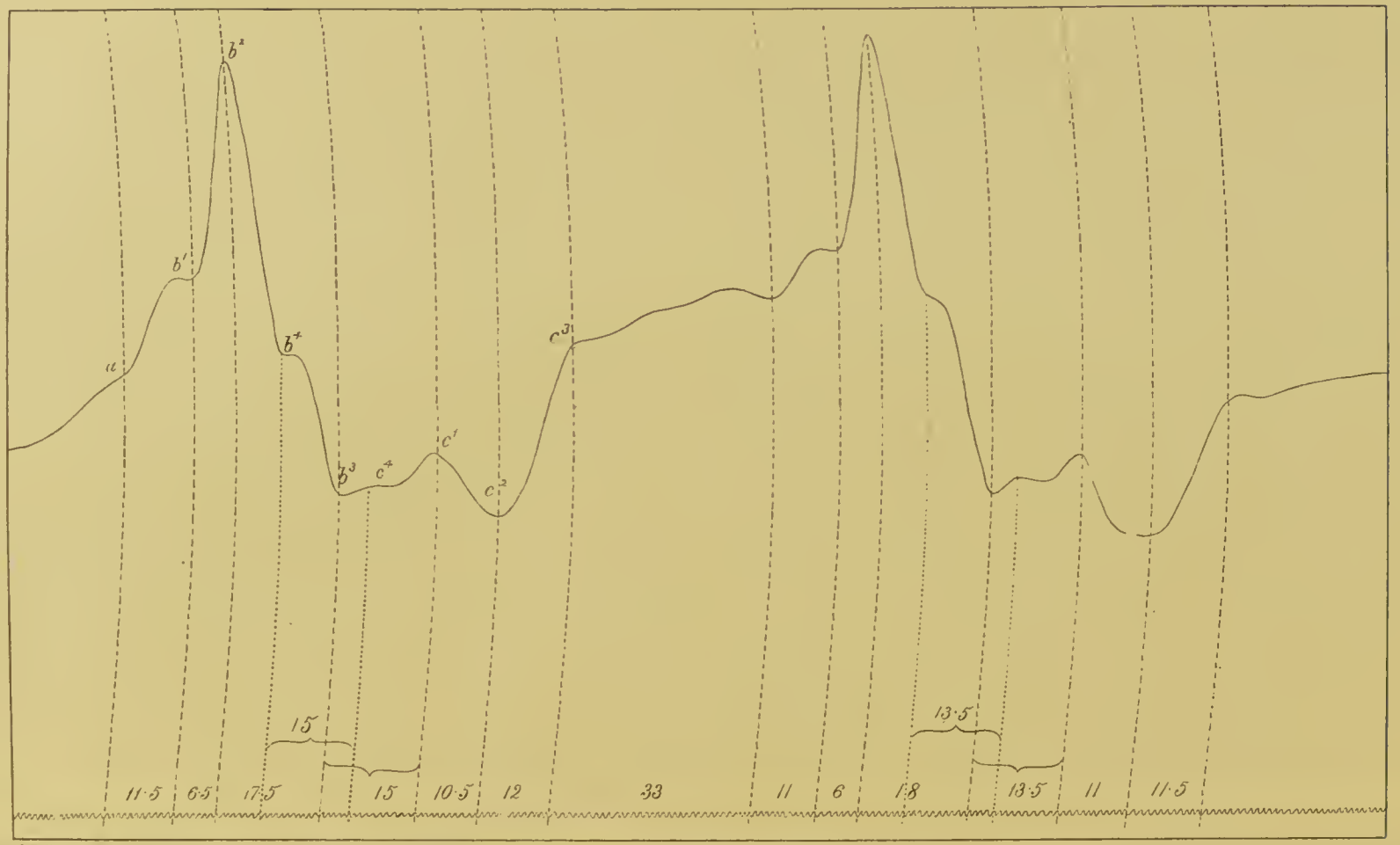
* Manual of the Physical Diagnosis of Diseases of the Heart. third edition, p. 121. 1881.

† *The Journal of Anatomy and Physiology*, Vol. xiv., p. 1. 1879.

‡ *Ibid.*, Vol. xiv., p 234. 1879.



W. Cathie Lith. Edin.

TRACING from CONUS ARTERIOSUS.

Part of one of these tracings, obtained by means of a direct cardiograph and a rapidly-revolving cylinder, registering also the vibrations of a tuning fork marking hundredths of a second, is reproduced in the accompanying plate, which is to be read from left to right. It is needless to give a description of the curve shewn by the tracing; all that it is necessary to mention is that *a* marks the beginning of auricular systole, b^1 the commencement of ventricular systole—coinciding with the beginning of the first sound—and c^1 the occurrence of the second sound. In the tracing given here, there is on the descending curve from b^2 to b^3 —which is caused by the emptying of the ventricles of the heart—a shoulder marked b^4; and there is also, in advance of c^1, which marks the incidence of the second sound, an elevation, c^4. On looking at the measured intervals of time recorded below the curve, the interesting fact will be noted that the period elapsing between b^3 and c^1 is precisely the same as that between b^4 and c^4. The conclusion is obvious that c^1 and c^4 represent the diastolic recoil on the sigmoid valves, while b^3 and b^4 are the terminations of ventricular emptying on the two sides. Many other tracings taken from the same case shew no shoulder in the position of b^4, and no elevation in the position of c^4; and it is further to be noted that on auscultation of the patient there never was any double second sound. In the latter of the two papers referred to, a laborious attempt was made to explain the facts, but curiously enough it was not remembered that when the tracings were taken the patient always held his breath. It is extremely probable that the respiration was suspended sometimes when in the phase of inspiration, and sometimes during the phase of expiration. If this were so there can be no difficulty in regarding the double wave of recoil taking place at the time of the second sound in many of the tracings as being by a physiological doubling.

The only other point to be alluded to in this place is a question as to which of the two second sounds occurs first.

One opinion, strongly urged by Balfour,* assumes that the closure of the sigmoid valves is accelerated on the side of the greatest pressure. This opinion has always appeared to me to charge nature with a defective provision for meeting excess of strain, and it flies in the face of our modern views as to compensation in cardiac disease. We know, as a matter of fact, how ample this provision is, and Cohnheim† has eloquently enlarged upon the impossibility of placing a limit upon it.

The other opinion—stoutly asserted by Barr,‡ and adopted by most of our recent writers, as, for instance, Bramwell,‖—holds that the second sound is later on the side which has most pressure in front of the ventricle, causing the ventricle therefore to be longer in overcoming the obstacle. This view is strictly in accord with what we know to be the inherent tendency of the cardiac mechanism, and it is also supported by clinical observation; for on careful auscultation of the aortic and pulmonary areas, in those cases of mitral disease which present the double second sound it is possible in every instance to determine that the first of the two sounds is louder in the aortic, and the second in the pulmonary area. This is an absolute demonstration that the second sound produced by the sigmoid valves at the pulmonary artery is later than that arising from the action of the aortic cusps. In other words, the right ventricle takes an appreciably longer time than the left to terminate its systole.

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* Clinical Lectures on Diseases of the Heart and Aorta, second edition, p. 33. 1882.

† Lectures on General Pathology, New Sydenham Society's translation, section i., p. 73. 1889.

‡ Liverpool Medico-Chirurgical Journal, No. 3, p. 195. 1882.

‖ Diseases of the Heart and Thoracic Aorta, p. 164. 1884.

Printed by Hall & English, No. 71, High Street, Birmingham.